colon was filled with blood. About 12 inches distal to the cecum in the ascending colon there was a fibrotic hemorrhagic tumor mass that was filled with large masses of clotted and fluid blood. On section of the mass, a diverticulum with acute inflammation and bleeding was noted. Microscopic examination confirmed the gross observations, and sections through the hemorrhagic mass showed ulceration of the mucosa of the diverticulum with severe, acute and chronic inflammatory changes extending through the entire thickness of the intestinal wall. Several blood vessels were seen at the base of the ulceration.

DISCUSSION

Burns[1] reported gross rectal bleeding in 42 of 118 cases of diverticular disease and pointed out that other lesions are often found at operation. In the series he reported, there were nine polyps and seven adenocarcinomas. He observed that in elderly persons gross hemorrhage may be the first sign of diverticular disease and that usually it can be abated with conservative management. In the series he reported, operation was not necessary in any case for the sole purpose of stopping bleeding.

Quinn described[5] a series of 49 patients treated in a 15-year period, all of whom had massive bleeding. All were treated with rest, sedation and transfusions. Five were operated on because of uncontrollable bleeding. Three who were not operated on died from massive bleeding.

The pathological findings in the case here reported emphasize the difficulty that a surgeon would have had in localizing the exact site of bleeding had the patient been in condition for exploration. In light of the known occurrence of associated lesions in many of these cases, careful study of the bowel before operation and examination of the entire colon at the time of operation are mandatory. One investigator said that if no bleeding is located at operation, subtotal colectomy—the cecum and entire colon to the rectosigmoid—may be indicated.[4]

SUMMARY

A case of extensive colonic diverticulosis and diverticulitis with massive fatal hemorrhage is reported. The circumstances emphasized the need for considering diverticulitis as a cause of gross bleeding and the difficulties that may be encountered in treatment.

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1. Burns, Francis J.: Bleeding diverticular disease of the colon, South. Med. J., 53:312, March 1960.

2. Delaney, William E., and Hedges, Richard C.: Acquired ilial diverticulosis and hemorrhagic diverticulitis, Gastro-enterology, 42:56, Jan. 1962.

3. Kelley, Donald J., Falk, Stuart, and Olson, Kenneth: Hemorrhage from a solitary jejunal diverticulum, Am. J. of Surg., 100:597, Oct. 1960.

4. Moos, Daniel L.: Massive colonic hemorrhage, Minn. Med., 44:41, Jan. 1961.

5. Quinn, William C.: Diverticular disease of the colon with hemorrhage—A study of 78 cases, Am. Surg., 26:171, March 1960.

6. Sheldon, William C., Lazar, Harold P., Richards, John W., and Henegar, George C.: Gastrointestinal hemorrhage from duodenal diverticula, Am. J. Digestive Diseases, 4:817 (New Series), October 1959.

Termination of Ventricular Tachycardia By External Countershock

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IN THE PAST YEAR, cardiac arrhythmias of ventricular and supraventricular origin resistant to intensive drug therapy have been successfully treated by external electric countershock.[1,3,6] A physician confronted with the problem of treating a patient with such an arrhythmia may, however, be reluctant to employ this therapy because of unfamiliarity and because of the paucity of reported cases so treated. Therefore, it is the purpose of this paper to report another case of persistent ventricular tachycardia, refractory to drugs, treated successfully by external electric countershock.

A 62-year-old man was admitted to Mount Zion Hospital and Medical Center for the treatment of uncomplicated acute myocardial infarction. On the morning of the tenth hospital day, he experienced crushing pain in the chest, and shock ensued. An electrocardiogram revealed ventricular tachycardia at a rate of 160 beats a minute. Pressor agents and antiarrhythmic drugs were given. At the end of the first day, the patient had received 1.4 gm of quinidine and 0.5 gm of procainamide. On the second day of the arrhythmia, he was given 2.4 gm of quinidine and it was necessary to substitute norepinephrine for metaraminol to maintain the blood pressure. On the third day, 1.2 gm of quinidine and 1 gm of procainamide were given and on the fourth day the amount of quinidine was increased to 3 gm. At this time, signs of heart failure were increasingly apparent and the blood urea nitrogen was 68 mg per 100 cc. On the fifth day, 4.2 gm of quinidine was given, yet the arrhythmia persisted at a rate of 160 to 140 beats per minute.

On the morning of the sixth day, a decision was made to attempt to terminate the ventricular tachycardia by electric countershock. The patient was anesthetized with intravenous sodium pentothal and nitrous oxide. Succinyl choline was also given.

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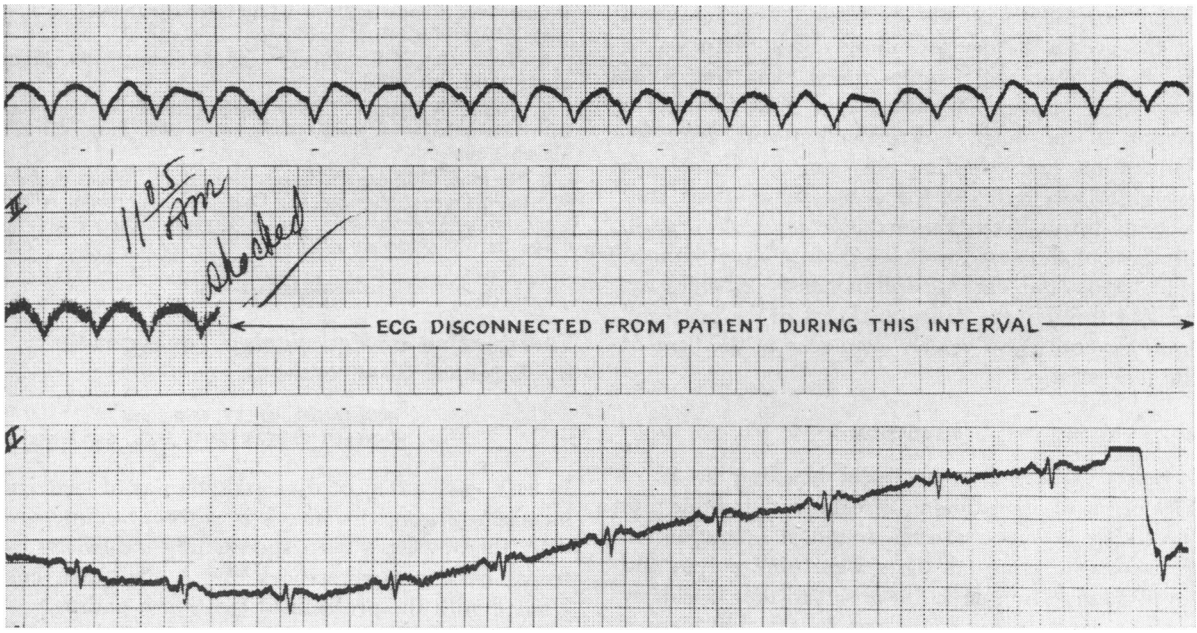

Figure 1.—Continuous lead II recording showing ventricular tachycardia and the reversion to sinus rhythm following electric countershock.

The electrical activity of the heart was observed with an electrocardiograph, and an external pacemaker was held in readiness for use in case of cardiac standstill. Conductive paste was applied to the electrodes and was rubbed into the skin only over the apical area of the heart and at the midright sternal border. The countershock electrodes, 8.5 cm in diameter, were then firmly placed on the two prepared areas. The cable from the electrocardiograph machine was disconnected from the patient just before the application of a single external countershock (440 volts, 60 cycle AC, 0.25 second duration). Immediately thereafter the machine was reconnected and the tracing revealed a sinus mechanism at a rate of 68 beats per minute (Figure 1). The blood pressure promptly rose from 90/60 to 170/90 mm of mercury and norepinephrine was reduced. When the patient awakened from anesthesia, clinical improvement was apparent and progressive. The blood pressure stabilized, pressor agents were discontinued within 24 hours, and the blood urea nitrogen returned to normal levels. Subsequent electrocardiograms demonstrated evolutionary changes of a myocardial infarction and convalescence was uneventful.

Electric countershock has been employed in the termination of ventricular fibrillation,[2,7] ventricular tachycardia[1,6] and supraventricular tachycardia.[3,6] When successful, its effect is both immediate and dramatic. It is well to point out that the nature of the underlying cardiac disease, the general anesthesia, and the possibility of ventricular fibrillation or standstill following electric shock, entail risks which must be carefully evaluated. Yet, this procedure may indeed prove to be a more valuable form of therapy than the administration of massive doses of toxic antiarrhythmic drugs.[4,5] In the present case it was promptly successful after failure to restore normal rhythm with drugs.

SUMMARY

Persistent ventricular tachycardia which followed an acute myocardial infarction was unsuccessfully treated with large doses of antiarrhythmic drugs for five days. The arrhythmia ended immediately following the application of external electric countershock.

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1. Alexander, S., Kleiger, R., and Lown, B.: Use of external electric countershock in the treatment of ventricular tachycardia, J.A.M.A., 177:916, 1961.

2. Kouwenhoven, W. B., Milnor, W. R., Knickerbocker, G. G., and Chestnut, W. R.: Closed chest defibrillation of the heart, Surgery, 42:550, 1957.

3. Paul M. H., and Miller, R. A.: External electrical termination of supraventricular arrhythmias in congenital heart disease, Circulation, 25:604, 1962.

4. Thomson, G. W.: Quinidine as a cause of sudden death, Circulation, 14:757, 1956.

5. Weisman, S. A.: Quinidine test dose: Is it warranted?, Dis. Chest, 35:428, 1959.

6. Zoll, P. M., and Linenthal, A. J.: Termination of refractory tachycardia by external countershock, Circulation, 25:596, 1962.

7. Zoll, P. M., Linenthal, A. J., Gibson, W., Paul, M. H., and Norman, L. R.: Termination of ventricular fibrillation in man by externally applied electric countershock, N.E.J.M., 254:727, 1956.